

(FILE 'HOME' ENTERED AT 17:33:16 ON 04 AUG 1998)

INDEX 'AGRICOLA, AIDSLINE, ANABSTR, AQUASCI, BIOBUSINESS, BIOSIS,
BIOTECHABS, BIOTECHDS, CABA, CANCERLIT, CAPLUS, CEABA, CEN, CIN,
CJACS, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DISSABS, DRUGB,
DRUGLAUNCH, DRUGNL, DRUGU, EMBAL, EMBASE, FSTA, GENBANK, ...'
ENTERED AT 17:33:28 ON 04 AUG 1998

SEA LIPASE(10W)UREASE

4 FILE ANABSTR
3 FILE AQUASCI
2 FILE BIOBUSINESS
34 FILE BIOSIS
12 FILE BIOTECHABS
12 FILE BIOTECHDS
6 FILE CABA
31 FILE CAPLUS
1 FILE CEABA
1 FILE DDFU
1 FILE DRUGU
6 FILE EMBASE
6 FILE FSTA
8 FILE IFIPAT
6 FILE LIFESCI
5 FILE MEDLINE
5 FILE SCISEARCH
6 FILE TOXLINE
2 FILE TOXLIT
48 FILE USPATFULL
15 FILE WPIDS
15 FILE WPINDEX

L1 QUE LIPASE(10W) UREASE

FILE 'USPATFULL, BIOSIS, CAPLUS, WPIDS, BIOTECHDS, IFIPAT, CABA,
EMBASE, FSTA, LIFESCI, TOXLINE, MEDLINE, SCISEARCH, ANABSTR,
AQUASCI, BIOBUSINESS, TOXLIT, CEABA, DRUGU' ENTERED AT 17:34:49 ON
04 AUG 1998

L2 201 S LIPASE(10W)UREASE

L3 141 DUP REM L2 (60 DUPLICATES REMOVED)

INDEX 'AGRICOLA, AIDSLINE, ANABSTR, AQUASCI, BIOBUSINESS, BIOSIS,
BIOTECHABS, BIOTECHDS, CABA, CANCERLIT, CAPLUS, CEABA, CEN, CIN,
CJACS, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DISSABS, DRUGB,
DRUGLAUNCH, DRUGNL, DRUGU, EMBAL, EMBASE, FSTA, GENBANK, ...'
ENTERED AT 17:39:44 ON 04 AUG 1998

SEA FAT ABSORPTION AND LIPASE

16 FILE AGRICOLA
3 FILE BIOBUSINESS
90 FILE BIOSIS
0* FILE BIOTECHABS
1 FILE BIOTECHDS
54 FILE CABA
2 FILE CANCERLIT
74 FILE CAPLUS
8 FILE CJACS
2 FILE DDFB

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0* FILE DDFU
7 FILE DGENE
2 FILE DISSABS
2 FILE DRUGB
1 FILE DRUGNL
24 FILE DRUGU
3 FILE EMBAL
101 FILE EMBASE
1 FILE FSTA
2 FILE IFIPAT
3 FILE LIFESCI
99 FILE MEDLINE
1 FILE NTIS
2 FILE PHAR
2 FILE PHIN
63 FILE SCISEARCH
10 FILE TOXLINE
16 FILE TOXLIT
34 FILE USPATFULL
5 FILE WPIDS
0* FILE WPINDEX
L4 QUE FAT ABSORPTION AND LIPASE
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FILE 'EMBASE, MEDLINE, BIOSIS, CAPLUS, SCISEARCH, CABA, USPATFULL,
DRUGU, AGRICOLA, TOXLIT, TOXLINE, CJACS, DGENE, WPIDS, BIOBUSINESS,
EMBAL, LIFESCI, CANCERLIT, DISSABS, DRUGB, IFIPAT, PHAR, PHIN,
BIOTECHDS, DRUGNL, FSTA, NTIS' ENTERED AT 17:49:37 ON 04 AUG 1998
L5 102 S FAT ABSORPTION(25W)LIPASE
L6 55 DUP REM L5 (47 DUPLICATES REMOVED)
L7 0 S L5 AND ANTIBOD?(25W)LIPASE

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INDEX 'AGRICOLA, AIDSLINE, ANABSTR, AQUASCI, BIOBUSINESS, BIOSIS,
BIOTECHABS, BIOTECHDS, CABA, CANCERLIT, CAPLUS, CEABA, CEN, CIN,
CJACS, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DISSABS, DRUGB,
DRUGLAUNCH, DRUGNL, DRUGU, EMBAL, EMBASE, FSTA, GENBANK, ...'
ENTERED AT 18:08:35 ON 04 AUG 1998
SEA TETRAHYDROLIPSTATIN AND OBESITY
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4 FILE BIOBUSINESS
4 FILE BIOSIS
0* FILE BIOTECHABS
SEA FAT REDUCTION AND PASSIVE IMMUNITY
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0* FILE BIOTECHABS
1 FILE CAPLUS
0* FILE DDFB
0* FILE DDFU

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FILE 'CAPLUS' ENTERED AT 18:20:19 ON 04 AUG 1998
L8 1 S FAT REDUCTION AND PASSIVE IMMUNITY

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FILE 'BIOBUSINESS' ENTERED AT 18:21:48 ON 04 AUG 1998
L9 4 S TETRAHYDROLIPSTATIN AND OBESITY

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INDEX 'AGRICOLA, AIDSLINE, ANABSTR, AQUASCI, BIOBUSINESS, BIOSIS,
BIOTECHABS, BIOTECHDS, CABA, CANCERLIT, CAPLUS, CEABA, CEN, CIN,
CJACS, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DISSABS, DRUGB,
DRUGLAUNCH, DRUGNL, DRUGU, EMBAL, EMBASE, FSTA, GENBANK, ...'
ENTERED AT 18:23:50 ON 04 AUG 1998
SEA LIPASE(10W)ANTIBOD?
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4 FILE AGRICOLA
7 FILE ANABSTR
2 FILE BIOBUSINESS
142 FILE BIOSIS

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0* FILE BIOTECHABS
 31 FILE BIOTECHDS
 18 FILE CABA
 6 FILE CANCERLIT
 135 FILE CAPLUS
 3 FILE CEABA
 2 FILE CJACS
 1 FILE CONFSCI
 1 FILE CROPB
 0* FILE DDFB
 0* FILE DDFU
 1 FILE DGENE
 5 FILE DISSABS
 1 FILE DRUGB
 1 FILE DRUGNL
 1 FILE DRUGU
 84 FILE EMBASE
 1 FILE FSTA
 3 FILE IFIPAT
 9 FILE JICST-EPLUS
 1 FILE KOSMET
 22 FILE LIFESCI
 92 FILE MEDLINE
 1 FILE PROMT
 48 FILE SCISEARCH
 4 FILE TOXLINE
 14 FILE TOXLIT
 38 FILE USPATFULL
 29 FILE WPIDS
 0* FILE WPINDEX
 L10 QUE LIPASE(10W) ANTIBOD?

FILE 'USPATFULL' ENTERED AT 18:36:12 ON 04 AUG 1998
 L11 63 S LIPASE(15W)INHIBIT? AND ANTIBOD?
 L12 5 S L11 AND LIPASE(25W)ANTIBOD?

WER 8 OF 24 AGRICOLA

AN 97:80298 AGRICOLA

DN IND20601809

TI Structure-function relationship of lipoprotein lipase-mediated enhancement of very low density lipoprotein binding and catabolism by the low density lipoprotein receptor. Functional importance of a properly folded surface loop covering the catalytic center.

AU Salinelli, S.; Lo, J.Y.; Mims, M.P.; Zsigmond, E.; Smith, L.C.; Chan, L.

CS Baylor College of Medicine, Houston, TX.

SO The Journal of biological chemistry, Sept 6, 1996. Vol. 271, No. 36. p. 21906-21913

Publisher: Bethesda, Md. : American Society for Biochemistry and Molecular Biology.

CODEN: JBCHA3; ISSN: 0021-9258

NTE Includes references

CY Maryland; United States

DT Article

FS U.S. Imprints not USDA, Experiment or Extension

LA English

AB We examined the structure-function relationship of human lipoprotein lipase (hLPL) in its ability to enhance the binding and catabolism of very low density lipoproteins (VLDL) in COS cells. Untransfected COS cells did not bind to or catabolize normal VLDL. Expression of wild-type hLPL by transient transfection enhanced binding, uptake, and degradation of the VLDL (a property of LPL that we call bridge function). Heparin pretreatment and a monoclonal **antibody** ID7 that blocks LDL receptor-binding domain of apoE both **inhibited** binding, and apoE2/E2 VLDL from a Type III hyperlipidemic subject did not bind. However, LDL did not reduce 125I-VLDL binding to the hLPL-expressing cells, whereas rabbit p-VLDL was an effective competitor. By contrast, LDL reduced uptake and degradation of 125I-VLDL to the same extent as excess unlabeled VLDL or beta-VLDL. These data suggest that binding occurs by direct interaction of VLDL with LPL but the subsequent catabolism of the VLDL is mediated by the LDL receptor. Mutant hLPLs that were catalytically inactive, S132A, S132D, as well as the partially active mutant, S251T, and S172G, gave normal enhancement of VLDL binding and catabolism, whereas the partially active mutant S172D had markedly impaired capacity for the process; thus, there is no correlation between bridge function and lipolytic activity. A naturally occurring genetic variant hLPL, S447 replaced by Ter, has normal bridge function. The catalytic center of LPL is covered by a 21-amino acid loop that must be repositioned before a lipid substrate can gain access to the active site for catalysis. We studied three hLPL loop mutants (LPL-CH, an enzymatically active mutant with the loop replaced by a hepatic lipase loop; LPL-CP, an enzymatically inactive mutant with the loop replaced by a **pancreatic lipase** loop; and C216S/C239S, an enzymatically inactive mutant with the pair of Cys residues delimiting the loop substituted by Ser residues) and a control double Cys mutant, C418S/C438S. Two of the loop mutants (LPL-CH and LPL-CP) and the control double Cys mutant C418S/ C438S gave normal enhancement of VLDL binding and catabolism, whereas the third loop mutant, C216S/ C239S, was completely inactive. We conclude that although catalytic activity and the actual primary sequence of the loop of LPL are relatively unimportant (wild-type LPL loop and **pancreatic lipase** loops have little sequence similarity), the intact folding of the loop, flanked by disulfide bonds, must be maintained for LPL to express its bridge function.

CC T200 Physiology of Human Nutrition
CT binding; catabolism; catalytic activity; cell lines; fibroblasts;
lipoprotein lipase; low density lipoprotein; man; molecular
conformation; mutants; protein degradation; receptors; stimulation;
targeted mutagenesis; uptake; very low density lipoprotein
ST cos cells
RN 9001-62-1 (HEPATIC LIPASE)
9001-62-1 (LIPASE)
9004-02-8 (LIPOPROTEIN LIPASE)
9005-49-6 (HEPARIN)

L

L6 ANSWER 17 OF 55 EMBASE COPYRIGHT 1998 ELSEVIER SCI. B.V.DUPLICATE
9

AN 91247714 EMBASE

TI The lipase inhibitor tetrahydrolipstatin binds covalently to the
putative active site serine of pancreatic lipase.

AU Hadvary P.; Sidler W.; Meister W.; Vetter W.; Wolfer H.

CS F. Hoffmann-La Roche Ltd., PF/CVD, 68/309, Grenzacherstrasse 124,
CH-4002 Basel, Switzerland

SO J. BIOL. CHEM., (1991) 266/4 (2021-2027).
ISSN: 0021-9258 CODEN: JBCHA3

CY United States

DT Journal

FS 029 Clinical Biochemistry
037 Drug Literature Index

LA English

AB Tetrahydrolipstatin (THL) is a selective inhibitor of **fat
absorption**. In animal models, it has anti-obesity and
anti-hypercholesterolemic activity and is presently in clinical
trials for these indications. THL binds covalently to pancreatic
lipase. Complete inhibition of lipolytic activity is
obtained concomitant with the incorporation of 1 mol of THL/mol of
enzyme. Pancreatic lipase is the best studied lipase, but published
results concerning its catalytic mechanism are still controversial.
In order to learn more about the inhibitory mechanism of THL, a
selective lipase inhibitor interacting at or near the catalytic
site, and therefore, to obtain more information on the catalytic
mechanism of lipase, we have determined the amino acid residue to
which THL is bound. After proteolytic degradation of porcine
pancreatic lipase inhibited with radioactively labeled THL, the
labeled peptides were isolated and analyzed by quantitative amino
acid analysis, N-terminal sequencing, and by mass spectrometry with
fast atom bombardment ionization. The data clearly show that THL is
bound as an ester to the serine 152 of the lipase.

AB Tetrahydrolipstatin (THL) is a selective inhibitor of **fat
absorption**. In animal models, it has anti-obesity and
anti-hypercholesterolemic activity and is presently in clinical
trials for these indications. THL binds covalently to pancreatic
lipase. Complete inhibition of lipolytic activity is
obtained concomitant with the incorporation of 1 mol of THL/mol of
enzyme. Pancreatic lipase. . .

9 ANSWER 78 OF 89 BIOSIS COPYRIGHT 1998 BIOSIS
 AN 80:190560 BIOSIS
 DN BA69:65556
 TI METABOLIC FUNCTION OF HEPARIN RELEASABLE LIVER LIPASE.
 AU JANSSEN H; VAN TOL A; HULSMANN W C
 CS DEP. BIOCHEM. I., MED. FAC., ERASMUS UNIV. ROTTERDAM, P.O. BOX 1738,
 3000 DR ROTTERDAM, NETH.
 SO BIOCHEM BIOPHYS RES COMMUN 92 (1). 1980. 53-59. CODEN: BBRCA9 ISSN:
 0006-291X
 LA English
 AB IV administration of specific [rabbit] antibody against
 heparin-releasable [rat] liver **lipase** (liver **lipase**
) induced a 75% inhibition of the enzyme activity in situ.
 Administration of the **antibody** resulted in an increase of
 high density lipoprotein (density range 1.050-1.13 g/ml; HDL2)
 phospholipid levels (20% after 1 h; 54% after 4 h). Short-term (1 h)
treatment with antibody had no significant effect on any of
 the other lipoprotein components. After long-term (4 h)
treatment the free cholesterol level of HDL2 and all
 components in the very low density lipoprotein (VLDL) + intermediate
 density lipoprotein (IDL) fraction were elevated (1.5-2.0-fold). In
 the low density lipoprotein (LDL) fraction only the phospholipid
 level was affected (increased by 72%). All lipid components in the
 HDL3 fraction were decreased by the antibody **treatment**, but
 this decrease was only statistically significant for the
 cholesteroesters. The removal rate of iodine-labeled high density
 lipoprotein (HDL) and LDL from serum was not affected by the antibody
treatment. Liver lipase may promote phospholipid removal in
 vivo. A lowering of liver lipase in situ apparently has profound
 consequences for serum lipoprotein metabolism.

L

L8 ANSWER 1 OF 1 CAPLUS COPYRIGHT 1998 ACS
 AN 1996:423154 CAPLUS
 DN 125:83777
 TI **Fat reduction** through the use of **passive immunity**
 AU Brodie, A.; Hu, C. Y.
 CS Department Animal Sciences, Oregon State University, Corvallis, 97331-6702, USA
 SO Biol. Fat Meat Anim. (1995), 70-77. Editor(s): Smith, Stephen B.; Smith, D. R. Publisher: American Society of Animal Science, Champaign, Ill.
 CODEN: 63AQA9
 DT Conference; General Review
 LA English
 CC 15-0 (Immunochemistry)
 AB A review with 34 refs. on use of **passive immunity** in relation to fat redn. in domestic meat-producing animals. Topics discussed include **passive immunity** against plasma membrane protein; **passive immunity** against growth hormone or somatostatin;.
 ST review fat redn meat animal immunity
 IT Adipose tissue
 (use of **passive immunity** to reduce fat in domestic animals)
 IT Animal
 (domestic, use of **passive immunity** to reduce fat in domestic animals)
 IT Immunity
 (passive, use of **passive immunity** to reduce fat in domestic animals)

9 ANSWER 45 OF 89 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 13

AN 89:423439 BIOSIS

DN BA88:81697

TI BIOSYNTHESIS OF LIPOPROTEIN **LIPASE** IN CULTURED MOUSE ADIPOCYTES I. CHARACTERIZATION OF A SPECIFIC **ANTIBODY** IN RELATIONSHIPS BETWEEN THE INTRACELLULAR AND SECRETED POOLS OF THE ENZYME.

AU VANNIER C; DESLEX S; PRADINES-FIGUERES A; AILHAUD G

CS EMBL, POSTFACH 110.2209, MEYERHOFSTRASSE 1, 6900 HEILDELBERG, FRG.

SO J BIOL CHEM 264 (22). 1989. 13199-13205. CODEN: JBCHA3 ISSN: 0021-9258

LA English

AB Polyclonal antibodies have been raised in rabbits against homogenous lipoprotein **lipase** (LPL) purified from the media of adipose 3T3-F442A cells. The **antibody** is able to inhibit the apolipoprotein C-II-dependent activity of LPL, to immunoprecipitate LPL under nondenaturing conditions from media and cellular extracts. A dot-blot immunoassay of secreted LPL is also described (range 0.1-0.7 melliunits). The secretion potential .mu., taken as the ratio of total releasable activity or antigen to initial cellular activity or antigen, was determined. This was shown in cells **treated** with heparin and cycloheximide to be equal to 1 for LPL antigen but significantly greater than 1 for LPL activity assayed under standard conditions. No LPL was actually degraded within the cells. A dramatic enhancement of the intracellular activity was induced by a mere dilution of detergent-**treated** cell lysates with no change in LPL antigen. The total intracellular activity reached a plateau at a value which now became identical to that obtained in the medium of cells exposed to heparin and cycloheximide. The existence of an inhibitor of LPL activity has been excluded as well as that of an increase in the catalytic activity of LPL during its secretion, before or after exposure to heparin. Our results indicate a systematic underestimation of LPL intracellular activity and suggest that LPL is present within intracellular cisternae in a cryptic state. This potetial activity can be fully unmasked in vitro. In agreement with other data (Vannier, C., and Ailhaud, G., (1989) J. Biol. Chem. 264, 13206-13216), our results appear to exclude the existence of a reservoir of catalytically inactive LPL molecules within adipose cells.

L

L12 ANSWER 4 OF 5 USPATFULL
 AN 90:59329 USPATFULL
 TI Dietary compositions and methods using bile salt-activated lipase
 IN Tang, Jordan J. N., Oklahoma City, OK, United States
 Wang, Chi-Sun, Oklahoma City, OK, United States
 PA Oklahoma Medical Research Foundation, Oklahoma City, OK, United
 States (U.S. corporation)
 PI US 4944944 900731
 AI US 87-122410 871119 (7)
 DT Utility
 EXNAM Primary Examiner: Stone, Jacqueline
 LREP Kilpatrick & Cody
 CLMN Number of Claims: 23
 ECL Exemplary Claim: 1
 DRWN No Drawings
 LN.CNT 586

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Dietary compositions, especially cow's milk-based infant formulas, are fortified with bile salt-activated lipase. Methods are provided for feeding newborn and premature infants which include administration of bile salt-activated lipase to increase fat digestion and therefore growth rate. Similarly, a method is provided to treat subjects for inadequate pancreatic enzyme production by administration of bile salt-activated lipase in conjunction with ingestion of fats.

=> d 112 4 kwic

L12 ANSWER 4 OF 5 USPATFULL
 SUMM As naturally occurring BAL has been isolated, anti-BAL **antibodies** may be produced and used to find the BAL clones in the expression libraries. Alternately, a partial structure of the. . .
 DETD . . . these enzymes had not been demonstrated. Accordingly, we next examined the cross-reactivity of human BAL and cat BAL by performing **antibody** inhibition studies.
 DETD **Antibodies** against human bile salt-activated **lipase** were prepared from a rabbit. The **antibodies** in the antiserum from the rabbit was collected and purified using affinity chromatography. Specifically, we used an affinity column loaded with covalently linked purified human BAL and Sepharose 4B. 3 M NaSCN was used to elute the retained **antibodies**. The monospecific **antibodies** then were used in a **lipase** assay procedure to test reactivity of the **antibodies** with human milk bile salt-activated lipase and with cat milk bile salt-activated lipase. The results are shown in Table II.

DETD TABLE II

EFFECT OF HUMAN MILK BAL **ANTIBODIES**
 ON BAL ACTIVITY IN HUMAN MILK,
 CAT MILK AND **ANTIBODY**-FREE SERUM

Control
 (Non-BAL immunized rabbit serum gamma globulin)
Antibody
 Aliquots % Activity
 (ml) Remaining CPM* BAL Activity **

0.000	100.0	2001	290.29
0.025	100.9	2019	292.90
0.050	99.6	1994	289.29
0.100	97.8	1957	283.91
0.150	98.0	1961	284.45
0.200	96.9	1938	281.15

Cat Milk

Antibody

Aliquots (ml)	% Activity Remaining	CPM*	BAL Activity**
0.000	100.0	2001	290.29
0.025	87.3	1747	253.44
0.050	74.5	1491	216.30
0.100	45.4	908	131.73
0.150	32.9	659	95.60
0.200	23.7	475	68.91

Human Milk

Antibody

Aliquots (ml)	% Activity Remaining	CPM*	BAL Activity**
0.000	100.0	993	144.06
0.025	41.1	408	59.19
0.050	15.0	149	21.62
0.100	5.4	54	7.83
0.150	5.1	51.	.

DETD As Table II shows, **antibodies** against bile salt-activated **lipase** from human milk **inhibited** enzyme activity in both cat milk and human milk. However, the human enzyme **antibodies** were only about 70% as reactive with the cat enzyme as with the human enzyme. From this we concluded that. . .